

### EPIDURAL STEROIDS: A LESSON UNLEARNT?

Despite the mounting evidence that epidural steroid injections (ESIs) are ineffective and carry significant risks, I am alarmed to read the following, gleaned from recent literature.

The UK based Health Technology Assessment (HTA) published in 2005( [\[1\]](#) ) concluded:

*"ESIs offer no sustained benefits to patients with sciatica in terms of pain, function or need for surgery. It may be concluded that lumbar ESIs have a weak, transient effect that is insufficient to provide a meaningful difference to patients in terms of functional improvement".*

A similar report in US in 2007 for the American Academy of Neurology( [\[2\]](#) ) concluded:

*"1) epidural steroid injections may result in some improvement in radicular lumbosacral pain when assessed between 2 and 6 weeks following the injection, compared to control treatments (Level C, Class I-III evidence).*

*The average magnitude of effect is small and generalizability of the observation is limited by the small number of studies, highly selected patient populations, few techniques and doses, and variable comparison treatments; 2) in general, epidural steroid injection for radicular lumbosacral pain does not impact average impairment of function, need for surgery, or provide long-term pain relief beyond 3 months.*

*Their **routine use for these indications is not recommended** (Level B, Class I-III evidence); 3) there is insufficient evidence to make any recommendation for the use of epidural steroid injections to treat radicular cervical pain (Level U)"*

. (My highlighting)

Note Level C Class I-III evidence corresponds to very weak recommendation, based on very low quality evidence.

Another recent paper, published in 2009 ( [\[3\]](#) ) by authors in Boston, US, rather alarmingly titled "*Epidural steroid injections are useful for the treatment of low back pain and radicular symptoms: pro*" notes:

*"Collectively, studies in acute radicular pain due to herniated nucleus pulposus have failed to show that epidural steroid injection reduces long-term pain or obviates the need for surgery. Similarly, there is scant evidence that epidural steroids have any beneficial effect in those with acute low back pain without leg pain or in those with chronic low back or leg pain."*

However, a recent paper from Seattle ( [\[4\]](#) ) suggests that: "*The clinical use of lumbar epidural steroid injections has increased dramatically*"

A paper in 2010 ( [\[5\]](#) ) reports that in 2006 alone, greater than 300,000 thoracolumbar transforaminal ESIs were performed on Medicare beneficiaries in the US.

### The risks:

A recent review ( [\[6\]](#) 2009) of the more popular 'transforaminal' epidural steroid injections, noted:

*"The most common and worrisome complications of transforaminal epidural steroid injections in the lumbar spine, though rare, are related to neural trauma, vascular trauma, intravascular injection, and infection".*

For example, the review cites a study by Botwin et al (2000) which had reported complications in 207 patients receiving 322 transforaminal lumbar epidural steroid injections.

Complications included transient headaches in 3.1%, increased back pain in 2.4%, increased leg pain in 0.6%, facial flushing in 1.2%, vasovagal reaction in 0.3%, increased blood sugar in 0.3%, and hypertension in 0.3%.

The incidence of minor complications was 9.6% per injection "with no major complications" (Note: cases of delayed onset such as arachnoiditis may not figure in this type of data).

However, there have been reported cases in the medical literature of major complications such as paraplegia (4) and even death.

Some of these complications can be due to vasospasm (particularly perhaps from the local anaesthetic that is usually injected with the steroid), direct vascular trauma, or embolus from particulate steroids.

Distal cord and conus injury can occur following transforaminal injections at lumbar levels, thought to be due to particulate corticosteroids causing embolisation in a radicular artery.

Looking at arachnoiditis, preservatives in the steroid preparations, especially polyethylene glycol (PEG) and benzyl alcohol, have been shown to be irritant to the arachnoid membrane, but there is also the issue of the invasive nature of the procedure and risk of dural puncture etc. There are a number of papers within the medical literature that concur with this (see my previous articles on [www.theaword.org](http://www.theaword.org) ).

There are 2 important recent animal studies looking at the effects of intrathecal administration of steroids. Note this is inside the dura, unlike epidurals, although there has been some evidence (I have discussed this in previous articles) that solution injected epidurally may end up inside the dura (i.e. intrathecal), especially if there is an unintended dural puncture, which is more likely in people who have scarring (e.g after spinal surgery) or any other abnormality of the dura.

Barros et al ( [7] ) looked at intrathecal betamethasone in dogs and found ***haemorrhage and necrosis and inflammatory infiltration in one dog as well as, in other two dogs, there was discreet fibrosis and thickness of the arachnoid layer which was focal in one and diffuse in the other.***

The authors concluded that these changes were due to the steroid.

Lima et al ( [8] ) looking at methylprednisolone injected intrathecally in dogs, found changes such as meningeal thickening and lymphocytic infiltrates in the blood vessels. In 3 animals, adhesion of pia, arachnoid, and dura matter was noted and the nerve roots were surrounded by fibrosis. In one animal, necrosis of the spinal cord was evident. They concluded:

***"The present study demonstrated that the intrathecal administration of commercially available methylprednisolone was responsible for causing histological changes in the spinal cord and meninges of the animals studied."***

(My emphasis)

The authors note that the study has some limitations: small number of sample, short period of follow-up and the use of a solution that is not preservative free. They point out that arachnoiditis has an insidious onset so 21 days might not be sufficient to detect the complete extent of the damage.

They note that their choice to use a preparation that was not preservative free was based on current clinical use and relevance, which might still be the case in Brazil and perhaps even in

Texas where some of the authors work. In the UK, preservative free solutions such as Celestone Soluspan® are in use, which should hopefully reduce the damage done by PEG etc.

### CONCLUSION:

Looking at the risk: benefit ration of epidural steroids, it seems hard to justify such widespread continued use of a treatment that not only fails to provide benefit, but carries with it significant risks of both minor and more major complications. We appear to have learnt nothing over the past decade.

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[1] available at: <http://www.hta.ac.uk/fullmono/mon933.pdf>

[2] available (short version) at: <http://www.neurology.org/content/68/10/723.short>

[3] abstract available at : <http://www.ncbi.nlm.nih.gov/pubmed/19126368>

[4] Friedrich & Harrast abstract available at: <http://www.ncbi.nlm.nih.gov/pubmed/20071921>

[5] Glaser, Pain Physician 2010 Root cause analysis of paraplegia following transforaminal epidural steroid injections: the 'unsafe' triangle. Available at see : <http://www.ncbi.nlm.nih.gov/pubmed/20495587>

[6] available at : <http://www.painphysicianjournal.com/2009/january/2009;12;233-251.pdf>

[7] abstract available at <http://www.ncbi.nlm.nih.gov/pubmed/17923956>

[8] abstract available at: <http://www.ncbi.nlm.nih.gov/pubmed/20859319>